WHAT IS CLAIMED IS:

1. A method of treating a patient in need of GTN therapy, comprising administering a therapeutically effective amount of GTN and of a therapeutically effective amount of an mtALDH inhibitor.

2. The method of Claim 1 where the patient has or is at risk for a stroke and the administration of the mtALDH inhibitor enables increasing the dosage of GTN.

3. The method of Claim 1 where the patient has a syndrome where NMDA receptor is overexcited and the administration of the mtALDH inhibitor enables increasing the dosage of GTN.

4. The method of Claim 3 where the syndrome is a neurodegenerative disorder, depression, AIDS dementia or CNS malignancy and the administration of the mtALDH inhibitor enables increasing the dosage of GTN.

5. The method of claim 1 wherein the mtALDH inhibitor causes postponement of GTN mediated tolerance.

6. The method of Claim 1 where the patient is in need of GTN preconditioning.

7. The method of Claim 1 where the patient has a disorder selected from the group consisting of ischemic coronary syndromes, severe peripheral vascular disease and transient ischemic attack.

8. The method of claim 1 where the inhibitor is a substrate that is competitive with GTN or agent which causes endogenous production of said substrate.

9. The method of Claim 8 where the substrate is acetaldehyde or agent which causes endogenous production thereof.

10. The method of Claim 1 where the inhibitor is a noncompetitive inhibitor.

11. The method of Claim 10 where the noncompetitive inhibitor is selected from the group consisting of cyanamide, chloral hydrate, acetaminophen, antibuse, disulfiram, or an oral hypoglycemic.

12. A method of increasing or stabilizing blood pressure in a hypotensive patient or in a patient sensitive to GTN mediation of hypotension or in a patient where hypotension is limiting, said patient being in need of GTN therapy, comprising administering to said patient a therapeutically effective amount of GTN and of an inhibitor of mtALDH.

13. The method of Claim 12 where the patient has a right ventricular infarct.

14. The method of Claim 12 where the patient has or is at risk for a stroke.

15. The method of Claim 12 where the patient is in need of GTN preconditioning.

16. The method of Claim 12 where the patient has myocardial ischemia.

17. The method of Claim 12 where the patient has portal hypertension.

18. The method of Claim 12 where the inhibitor is a substrate that is competitive with GTN or agent that causes endogenous production of said substrate.

19. The method of Claim 18 where the inhibitor that is competitive with GTN is acetaldehyde or agent that causes endogenous production of acetaldehyde.

20. The method of Claim 12 where the inhibitor is a noncompetitive inhibitor.

21. The method of Claim 20 where the noncompetitive inhibitor is chloral hydrate, cyanamide, antibuse, acetaminophen, disulfiram, or oral hypoglycemic.

22. A method of treating ischemia or congestive heart failure in a patient in need thereof, comprising administering to said patient a therapeutically effective amount of a nitrite generator which is not GTN and is targeted to mitochondria.

23. The method of Claim 22 where the nitrite generator is a nitro derivative of a lipophilic and/or cationic thiol or a nitro derivative of a lipophilic and/or cationic alcohol.

24. A method of potentiating the effect of organic nitrate in a patient administered organic nitrate, comprising administering to said patient

therapeutically effective amount of the organic nitrate and also a therapeutically effective amount of a mitochondria-selective thiol or of a transgene of *mtALDH* which overcomes inhibition of endogenous mtALDH and/or increases NO bioavailability and/or causes decrease in GTN tolerance

25. The method of Claim 24 where the organic nitrate is GTN and mitochondria selective thiol is administered to potentiate the effect of the GTN by reversing GTN tolerance caused by oxidation by GTN of mtALDH by reducing the oxidized mtALDH.

26. The method of Claim 24 where the organic nitrate is GTN and mitochondria selective thiol is administered to potentiate the effect of GTN by improving the efficacy or increasing the potency of the GTN by reducing and thereby activating mtALDH or oxidized mtALDH and also by facilitating removal of $NO_2$ groups.

27. The method of Claim 24 where the thiol is positively charge.

28. The method of Claim 27 where the thiol contains a thiobutyldiphenylphosphonium cation.

29. The method of Claim 24 where the transgene of *mtALDH* is administered and administration is by infusion through a catheter inserted into a coronary artery.

30. The method of claim 24 where the patient is affected with restenosis.

31. The method of Claim 24 where the patient is affected with an unstable coronary syndrome.

32. The method of Claim 31 where the unstable coronary syndrome is unstable angina.

33. The method of Claim 24 where the patient is affected with asthma.

34. The method of Claim 24 where the patient is affected with rectal spasm.

35. A method of treating a patient in need of nitroglycerin therapy comprising administering to such patient a therapeutically effective amount of nitroglycerin and a therapeutically effective amount of a dithiol and/or a therapeutically effective amount of other reductant capable of activating mtALDH.

36. The method of Claim 35 where the patient is affected with a disorder selected from the group consisting of unstable coronary syndromes, restenosis, heart failure, asthma and rectal spasm.

37. The method of Claim 36 where dithiol is administered to reverse or postpone or prevent nitroglycerin tolerance from occurring in the patient.

38. The method of Claim 37 where the dithiol is a mitochondria selective dithiol.

39. The method of Claim 38 where the mitochondria selective dithiol is dihydrolipoic acid.

40. The method of Claim 37 where reductant capable of activating mtALDH is administered to reverse or postpone or prevent nitroglycerin tolerance from occurring in the patient.

41. The method of Claim 40 where the reductant is tris(2-carboxyethyl-phosphine).

42. A method of countering loss of GTN activity in a patient administered GTN, comprising administering to said patient a drug imparting NO bioactivity that does not require mtALDH for its metabolism.

43. The method of Claim 42 where the drug is a glycerol mononitrite or a glycerol thionitrite or a glycerol thionitrate.

44. The method of Claim 42 where the patient is in need of antianginal or preload reducing activity.

45. A method of treating a patient in need of nitrosovasodilation, comprising administering to the patient a nitrosovasodilator which is not a substrate for human mtALDH and/or is a substrate for a different human ALDH enzyme, to avoid tolerance, mitochondrial dysfunction and atherogenic potential.

46. The method of Claim 45 where nitrosovasodilator is a substrate for a different human aldehyde dehydrogenase enzyme.

47. The method of Claim 45 wherein the nitrosovasodilator substrate for human ALDH enzyme which is not human mtALDH contains a nitro group.

48. The method of Claim 45 where the patient is in need of antianginal or preload reducing activity.

49. The method of Claim 48 where the nitrosovasodilator is 2-glycerol mononitrite.

50. A method of treating a patient in need of antianginal and/or preload reducing activity, comprising administering to said patient a therapeutically effective amount of a mitochondria impermeable nitrate or nitrite.

51. The method of Claim 50 where nitrate or nitrite is negatively charged.

52. A method of determining cross-tolerance of nitroglycerin and other drug comprising assaying to determine whether the other drug inhibits mammalian mtALDH with inhibition indicating cross-tolerance.

53. The method of Claim 52 where the other drug is a drug containing an $NO_x$ group where x is 1 or 2 or which is metabolized to produce NO bioactivity.

54. A method of selecting between nitroglycerin and another antianginal or preload reducing drug for administering to a patient in need of antianginal and/or preload reducing activity, comprising determining whether polymorphism exists in the mtALDH gene of the patient and administering GTN if not, and other antianginal or preload reducing drug if so.

55. The method of Claim 54 where the drug administered if polymorphism exists is a nitrate ester different from GTN.

56. A method of determining dose of nitrate for a patient comprising purifying mtALDH from the patient and determining the activity of the purified mtALDH on the nitrate to determine dose that is effectively metabolized and does not cause inactivation of mtALDH.

57. The method of Claim 56 where the nitrate is to be given prophylactically to prevent angina.

58. The method of Claim 57 where the determination is used as a predictor in respect to tolerance.

59. The method of Claim 57 where the mtALDH purified is from erythrocyte or other blood cell of said patient.

60. Composition for intravenous administration of nitroglycerin, said composition comprising a therapeutically effective amount of nitroglycerin in a carrier and not containing compound which is or metabolizes to substrate competitive with nitroglycerin as a substrate for mitochondrial aldehyde dehydrogenase, or containing as the only carrier, one which contains no more than 25% ethanol.

61. A composition as claimed in Claim 60 which does not contain ethanol.

62. A composition as claimed in Claim 61 where the carrier comprises vehicle selected from the group consisting of a glycol, glycerol, an alcohol different from ethanol, saline and phosphate buffered saline.

63. A composition as claimed in Claim 62 where the carrier is composed of propylene glycol and water.

64. A composition as claimed in Claim 62 where the carrier is composed of 50% propylene glycol in water.

65. A composition as claimed in Claim 62 where the carrier is pure saline.

66. A composition as claimed in Claim 60 where the composition contains as the only carrier one which contains from 0.1 to 25% ethanol.

67. A composition as claimed in Claim 60 which does not contain an alcohol.

68. A composition as claimed in Claim 60 which is completely devoid of alcohol.